EARDRUM DISPLACEMENT FOLLOWING STAPEDIUS MUSCLE CONTRACTION

by

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SUMMARY PAGE

THE PROBLEM

To determine whether stapedius muscle contraction leads to eardrum displacements and to further understand the dynamics of any movements which occur.

FINDINGS

Simultaneous electronic monitoring of: (1) acoustic impedance, and (2) air pressure in the closed ear canal allowed differentiation of the actions of the tensor tympani and stapedius muscles. Extensive control procedures were used to isolate muscle contractions and their effects. The stapedius has a less predictable action on the eardrum than the tensor tympani. Extremely small biphasic and monophasic movements of the eardrum occurred to stapedius-only contraction. When the tensor tympani was also active, its effects over-rode those of the stapedius, causing relatively large inward or biphasic displacements of the eardrum. In the tensor-only ear, much larger inward-only eardrum movements were seen, indicating that the presence of the stapedius muscle modulates eardrum movement caused by tensor contraction. Estimates of maximum eardrum displacement were calculated based on a model of the external ear canal and eardrum. This movement amounted to about 4.1×10^{-4} mm.

APPLICATION

For the use of medical and bioengineering personnel interested in using the stapedius reflex as a protective device in a hearing conservation program.

ADMINISTRATIVE INFORMATION

This investigation was conducted as part of Bureau of Medicine and Surgery Research Work Unit MF51.524-9010DA5G. The present report was submitted for publication on 27 August 1972. It is Report No. 21 on the indicated Work Unit and has been designated as Naval Submarine Medical Research Laboratory Report No. 723.

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ABSTRACT

Simultaneous monitoring in human subjects on the same ear of eardrum displacement by tympanomanometry, and acoustical impedance by the Madsen bridge, provided information concerning contraction of the stapedius muscle and its effect on eardrum displacement. Extensive control procedures were employed to elicit only the stapedius reflex: lower intensity auditory stimulation, electrocutaneous stimulation of the homolateral external ear canal, and anesthetization of nerves leading to the tensor tampani. 1. Extremely small biphasic and monophasic eardrum movements were seen in the stapedius-only ear to auditory and electrocutaneous stimulation; the form of the response was much less predictable to auditory stimulation. 2. At higher sound intensities relatively large inward and biphasic movements of the eardrum occurred in the normal ear, unquestionably due to contraction of the tensor tympani. These results were further validated in a group of stapedectomized ears, without the stapedius but with normal tensor tympani. 3. Biphasic responses did not occur in the tensor tympani-only ear, only monophasic inward responses. 4. Upon air-jet stimulation to the orbit of the eye, these subjects had an accentuated tensor response in that large inward movements of the eardrum occurred as compared to those in normal ears, suggesting that there is an alteration of the tensor response by the presence of the stapedius muscle. Estimates of the actual eardrum displacements were calculated based on a model of the external ear canal and eardrum.

EARDRUM DISPLACEMENT FOLLOWING STAPEDIUS MUSCLE CONTRACTION

INTRODUCTION

Hensen in 1878 first observed contraction of the middle ear muscles in dogs upon exposure to an intense acoustic stimulus[1]. Since that time the properties of the middle ear muscle reflexes have been extensively studied, and a sizable literature has accumulated on the subject.

The aims of previous investigators differed widely: some have studied the importance of the reflexes for controlling the sound transmission through the middle ear, others have studied physiological aspects, while others have simply used the reflexes for studies of the physical properties of the middle ear mechanism.

Many unresolved questions remain. For only one example, there are conflicting reports of eardrum movement following stapedius muscle contraction. This study investigates this particular area in the human ear, utilizing more complete controls than used heretofore.

Anatomy

The middle ear possesses two small muscles, the stapedius and the tensor tympani. The stapedius was first described by Varolius in 1591 and the tensor by Eustachius in 1564. The mean length of the human stapedius is 6.3 mm with a cross section of 4.9 sq. mm. Mean length of the human tensor tympani is 25 mm, with a cross section of 5.85 sq. mm. (reference 1).

The stapedius muscle occupies a very small cavity on the posterior wall of the tympanic cavity; its tendon passes through an aperture, turns a little downwards, and proceeds medially to the posterior margin of the head of the stapes close to the articulation with the lenticular process of the incus. On contraction, the stapes is pulled in a downward and outward direction[2].

The tensor tympani lies in a bony canal above the pharyngotympanic tube; its tendon runs through a bony channel, makes a turn around a hook on the promontory called the cochleariform process, and enters the middle ear cavity where it inserts on the medial side of the manubrium of the malleus. The cochleariform process changes the direction of action from anterior to anteromedial[3].

Innervation

The stapedius is innervated by a branch of the facial nerve[4], the tensor tympani by the mandibular branch of the trigeminal nerve through the otic ganglion[5]; there is also a connection with the tympanic plexus[6] and one with the sphenopalatine ganglion through the large deep petrosal nerve[7].

Eardrum Movement: Review of Literature

Ostmann in 1898 from visual observations concluded there was an inward movement of the eardrum during the

acoustic reflex, but Waar in 1923 and Lüscher in 1929 found no such movement during the reflex[1] tensor contraction in the rabbit to auditory stimulus, but at that date no recording system was sensitive enough objectively to register movements.

Kohler (1909)[1] and later Kobrak (1957)[8] used a very small reflecting mirror cemented to the eardrum by which small movements of the drum could be recorded. Using an acoustic stimulus, Kobrak reported movements in angle of rotation and umbo displacement.

Mangold (1913)[9] first succeeded in recording pressure changes in the human ear canal; in one of several subjects presumed able to contract the tensor voluntarily, eardrum movement was recorded.

Terkildsen (1957, 1960)[10,11] with a more sensitive manometric method found total displacement of the tympanic membrane minute movement to be only a little less than 1 cu. mm. This accounts for unsuccessful attempts to visualize these movements by direct observation of the eardrum.

Terkildsen demonstrated outward movement of the eardrum in response to 1 kHz at 100 dB to the contralateral ear in over half of the 60 ears tested, attributing these outward movements to the stapedius. Some inward movements of the eardrum were seen which he attributed to the tensor.

Mendelson (1957)[12] first successfully used an electrical pressure manometer to record pressure changes in a sealed ear canal; volume displacements of the order of 0.1 cu mm were recordable. His results were at best ambiguous. Using an auditory stimulus, he obtained little consistency in response; some eardrum movements were inward, some outward, and a very few biphasic. The response patterns also varied.

Weiss et al. (1962)[13] using a very sensitive electric pressure transducer and a signal average provided extensive information concerning eardrum movements in response to sound. Patterns varied from subject to subject; in some the direction of movement was inward, while in others it was outward.

Holst et al. (1964)[14] used sensitive recording methods and three different types of stimuli, (a) tactile stimulation of the orbital region by air blowing, (b) tactile stimulation of the contralateral ear, and (c) acoustic stimulation of the contralateral ear with pure tones, usually 500 Hz at 127 dB SPL. The subjects used included both normal and pathological ears. His results with normal ears indicated monophasic inward, monophasic outward, and biphasic movements of the drum. Outward movements occurred mainly during blowing against the ear and acoustic stimuli. Biphasic responses were about equally distributed in the different stimuli. Patients with chronic otitis were examined and none gave a recordable response to any stimuli used. Two patients with a broken ossicular chain were examined and inward movements with air to the eye were found.

Moller (1964)[15] attempted the first rigorous control in studying the middle ear muscles using anesthetized cats and

rabbits. Movements of the eardrum, acoustic impedance, and the cochlear microphonic were monitored on the same ear. Muscle tendons were selectively cut to eliminate muscle action. The muscles were electrically stimulated with bipolar electrodes near the origin of the tensor tympani and near the tendon of the stapedius muscle. Contraction of the tensor muscle produced a decrease in pressure in the ear canal associated with an inward movement of the drum. Contraction of the stapedius muscle did not produce any significant movement of the eardrum compared to that produced by the tensor. Changes in the acoustic impedance at the eardrum and changes in the cochlear microphonic were quite similar. A contraction of the stapedius muscle produced a substantial impedance change with little air pressure change. The tensor-only contraction, however, produced quite a notable change in air pressure as well as a change in acoustic impedance.

Purpose

It was hoped that a sensitive measurement of eardrum displacement, with selective elicitation of tympanic muscle responses, together with simultaneous recording of acoustic impedance, could provide information making possible a differentiation between the actions of the two muscles in the human. This study concentrated on eardrum movement following stapedius-only contraction.

Inasmuch as the only study employing really adequate controls on eardrum movement as effected by the middle ear muscles, that of Moller, was done on rabbits, and in view of the wide inter-species differences known to exist in middle ear muscle activity, it was desired to duplicate in the human ear a portion of Moller's study.

PROCEDURE

Stapedius-Only Stimulation

(a) Lower SPL

Eliasson and Gisselsson (1955)[16] found that the stapedius threshold in cat was 30 dB lower than the tensor. Holst et al. (1964)[14] noted that the threshold for stimulating the tensor is about 15-20 dB higher if the stapedius has been sectioned. Metz (1952)[17] found that the acoustic threshold of the stapedius was in the range of 70-90 dB SPL.

Feldman (1967)[18] on patients with a sectioned stapedius tendon and others with a sectioned tendon of the tensor tympani, found no impedance change to noise from tensor-only contraction.

(b) Slight Electric Shock to Homolateral Ear

Klockhoff and Anderson (1960)[19] and Klockhoff (1961)[20] used a surface electrode applied in the auditory canal; subjects perceived the sensation as paresthesias, which did not cause pain or discomfort. The authors successfully demonstrated that this indeed was a homolateral stapedius reflex.

Klockhoff (1961)[20] showed that the stapedius reflex to shock is absent during conductive impairment (otosclerosis,

otosalpingitis with transudation, adhesive otitis), and in patients with unilateral facial palsy in whom the stapedius muscle was ascertained to be inoperative. To demonstrate that contraction of the stapedius muscle was due to a reflex and not consequent upon direct electrical stimulation of the muscle, the ear canal was anesthetized, whereupon no electrocutaneous reflex existed although the acoustic reflex remained unchanged.

(c) Tensor Anesthetized

Clubb (1965)[7] used a cocaine solution to block the tensor muscle. He found in patients with retraction of the eardrum that it would resume its normal position after the sphenopalatine ganglion had been packed with cotton saturated in cocaine.

(d) Tensor-Only Stimulation

Patients were assembled with stapedectomized ears, in whom the stapedius had been sectioned during the surgical procedure for otosclerosis. It was hypothesized that any eardrum movements which occurred during stapedius-only experimentation would logically be absent in this group.

Pressure Recording

Apparatus

A Sanborn Model 270 gas pressure transducer was used to measure volume displacements in the ear canal according to the practice in the NAVSUBMEDRSCHLAB (K. G. Wing, Unpubl. results). When this strain-gage manometer is connected via a short, small plastic tube to the external ear canal and sealed, a closed cavity is formed. Only one connection is made to the manometer. The other is left open and consequently the device indicates a differential pressure. Changes, if any, in eardrum position are transformed to pressure changes in the closed cavity, and transduced to an electrical signal, amplified, and led to one pen of a Beckman two-channel recording Dynagraph.

Previous research on eardrum movement during middle ear muscle contractions indicated biphasic movements. It was felt necessary to determine if there was a distortion produced in the transducer which would permit monophasic responses to appear biphasic. A plastic tube with a stopcock valve was attached to one leg of the differential transducer. A pressure of 1 mm H_2O was put into the tube. The stopcock was then opened as quickly as possible. The pressure vs time function was written by the Beckman Dynagraph (see Fig. 1). It was concluded that any pressure changes in the external canal would be a faithful indication of eardrum movement.

Impedance Recording

The electroacoustic impedance bridge first developed by Metz (1946)[21] and later manufactured by the Madsen Company allows an accurate measurement of the impedance at the eardrum.

Figure 2 depicts the principle components used in the electroacoustic measurement of impedance. A probe

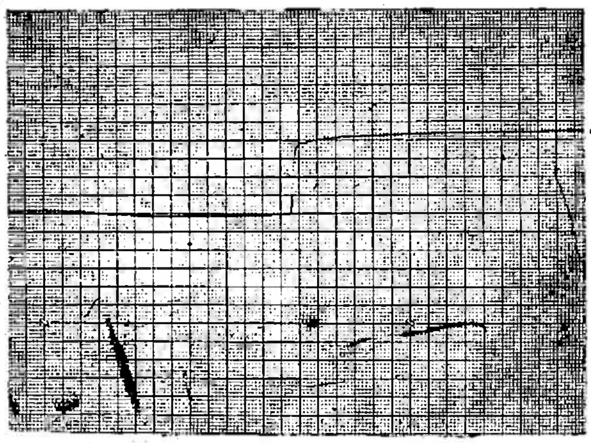

Fig. 1. The pressure vs time function of the pressure transducer. Damping was sufficient in the transducer to eliminate an overshoot.

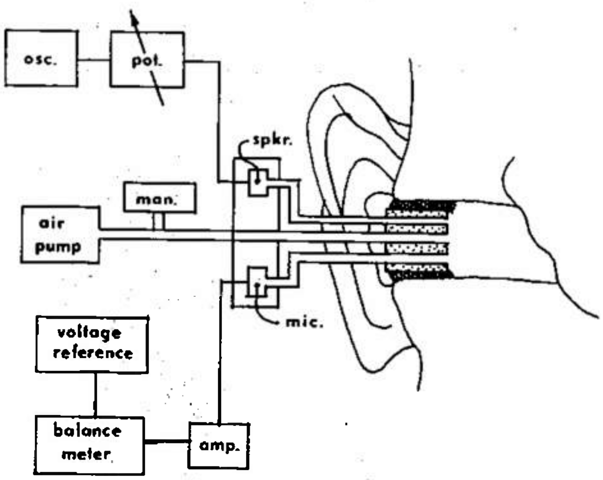

Fig. 2. Block diagram illustrating the operation of the Madsen Model Z0-70 electroacoustic impedance bridge.

tube is sealed into the external ear canal and forms a closed cavity, the bounds being the eardrum, the inner walls of the meatus, and the surface of the probe tip. Three tubes are led from the probe tip. One tube is used to deliver a probe tone of 220 Hz to the closed cavity. The oscillator is contained in the bridge.

The second tube is connected to a microphone that is used to measure the SPL in the closed cavity.

The third tube is ordinarily connected to a pressure manometer and air pump. Fig. 3 notes that in this study this tube led instead to the Sanborn 270.

What the electroacoustic impedance bridge actually measures is small changes in the SPL in the ear canal. When the stapedius muscle contracts, the attenuation it causes is reflected by a decrease in the equivalent volume in the closed cavity. Consequently, there is an increase in the intensity of SPL in the cavity. The output is then led to the other pen of the Beckman Dynagraph.

The unique feature of the apparatus used here was the simultaneous recording of both pressure and impedance changes in the same ear.

Stimulus Systems

Eight normal-hearing persons were each given three types of stimuli. These

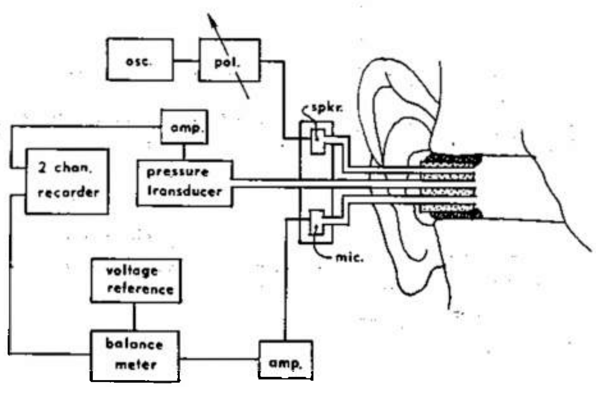

Fig. 3. Block diagram illustrating the modified apparatus used for simultaneously recording acoustical impedance and eardrum movement.

were NAVSUBMEDRSCHLAB staff members, enlisted Navy men, and graduate students at University of Connecticut.

(a) Auditory to Contralateral Ear

An audio oscillator set to 1 kHz was interrupted with an electronic timer and switch, led to an adjustable attenuator (0-100 dB), and the output connected to a Permoflux, PDR-600, earphone fitted with an MX-41 AR cushion. A voltmeter was used to fix the SPL in a 9A coupler at the calibrated earphone (see Fig. 4).

The intensity progressed from 70 dB to 115 dB SPL with increments of 5 dB. The duration of stimulus was 750 msec with about 1 sec. between each occurrence. The rise-fall time characteristic of each acoustical pulse was 40 msec. Simultaneous monitoring of both the impedance and the eardrum movement were maintained throughout the presentation of stimuli.

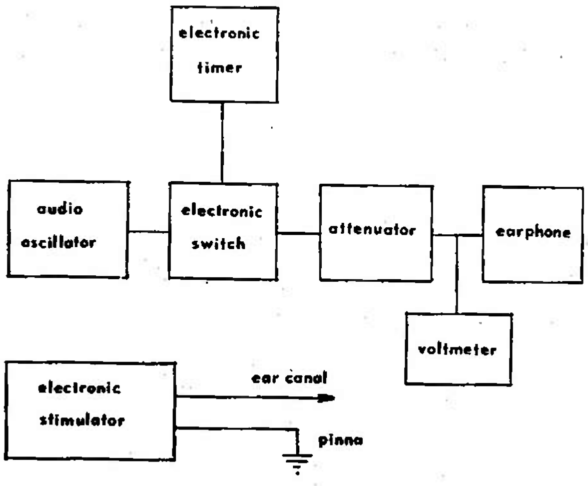

Fig. 4. Block diagram illustrating the stimulus delivery system.

(b) Electrocutaneous to Homolateral Ear

A Grass Model 11 electrical stimulator led to two electrodes attached to the subject. One electrode (see Fig. 5) was in contact to the Model ZO-70 probe tip. Tinsel wire having a clothlike consistency was carefully spread around the tip of the probe. The ground electrode was attached to the pinna with an electrode clip. Interruption of the electrical stimulus was accomplished by a switch located on the stimulator.

Once the electrodes were attached, short D.C. electrical pulses about 1 sec. in duration were applied to the canal. The voltage was increased until a good stapedius reflex (indicated by

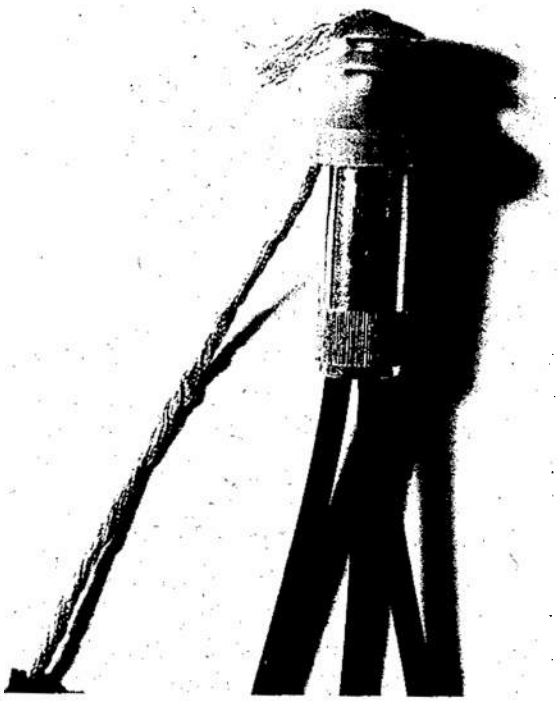

Fig. 5. The modified probe tip showing the electrode wire that makes contact with the inside of the external ear canal.

impedance monitoring) was obtained, providing it was not painful to the subject. Simultaneous monitoring of impedance and eardrum movement were maintained throughout the presentation of stimuli.

(c) Air-Jet to Orbit

Figure 6 is a photograph of the apparatus used to provide an air-jet to the orbit of the eye. The helmet and positioner held the air tube a constant distance and was comfortable for the subject. Squeezing the rubber bulb provided a current of air adjustable in strength.

Clinical Material

Stapedius-Only Ear

An otologist (Dr. Frederick L. Dey, New London, Conn.) anesthetized the sphenopalatine ganglion in three NAVSUBMEDRSCHLAB staff members. This procedure should block contraction of the tensor muscle. While the subject was seated in a chair an otologist inserted a nasal rod into the nasal opening on the side of the test ear. The end of the nasal rod was packed with cotton that was saturated with a 50% cocaine-hydrochloride solution. The time of insertion varied with the responses ob-

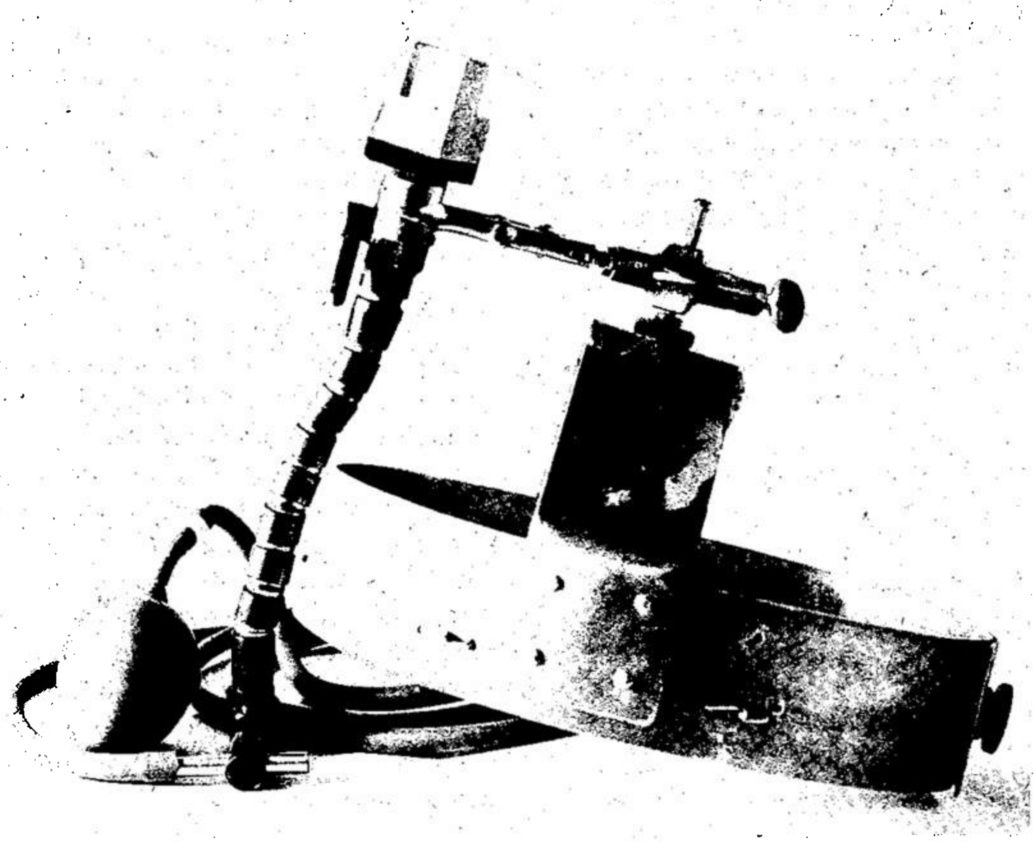

Fig. 6. The apparatus used to deliver an air-jet to the orbit of the eye.

tained. Acoustic stimuli only were used. Simultaneous monitoring of impedance and eardrum movement were maintained during and after cocainization.

Tensor-Only Ear

Two stapedectomized volunteers (3 ears) from Dr. Dey's practice provided a unique control in that they possessed only an intact tensor muscle. All three types of stimuli were used.

RESULTS AND DISCUSSION

A fuller account may be found in[22].

The lack of a stimulus-marking pen on the two-channel recorder necessitated the use of a latency measure to describe the approximate onset of the stimulus. This was easily accomplished using the same two-channel recorder. The latency of the stapedius is indicated in Fig. 7. The contraction of the muscle was measured using the impedance technique and is displayed on

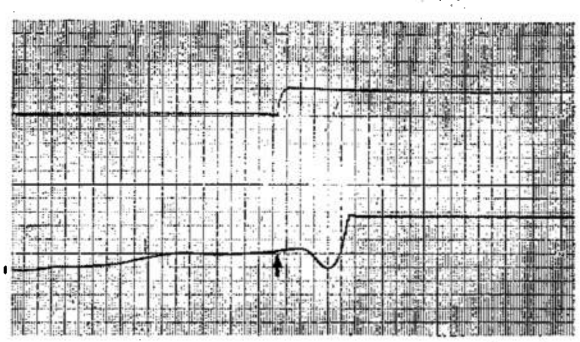

Fig. 7. Latency of the stapedius reflex. The top tracing is used for marking the onset of the stimulus (1 kHz, 90 dB SPL). The lower tracing indicates impedance.

the lower tracing. An auditory stimulus of 1 kHz at 90 dB SPL was delivered to the contralateral ear. The upper tracing is a very precise mark of the stimulus onset. The results indicate a latency of 64 msec. This is in close agreement with the data of Metz (1951)[23], who found using impedance change recordings that the latency of the stapedius reflex in human subjects varied from 35-150 msec. depending on the intensity of the stimulus. Using the time interval of 64 msec, stimulus onset markings were manually placed onto the graphs and are indicated by an ↑.

Experiment I: Auditory Stimuli

Table I summarizes the results for the eight subjects. Only one person failed to indicate any movement of the eardrum to any of the stimulus intensities. All movements in this experiment were monophasically inward. The mean threshold and standard deviation are in general concurrence with the data presented by Metz (1946)[21] and more recently by Deutsch (1968)[24]. It was hypothesized that an intensity 5 dB above threshold for the stapedius reflex would be an appropriate level to look at stapedius-only contraction. When this level was used, there were no subjects that indicated eardrum displacement. Excluding one subject that did not indicate any eardrum displacement, the difference between indications of impedance change and eardrum displacement were between 10 and 30 dB. The mean eardrum displacement threshold occurred at 100.0 dB with a standard deviation of 5.77.

Table I. Impedance and eardrum displacement thresholds (1 kHz)

SUBJECT	IMPEDANCE (SPL)	DISPLACEMENT (SPL)
1	75	105
2	90	110
3	85	95
4	85	95
5	75	95
6	85	100
7	80	NR
8	80	100
	Mean 81.9	Mean 100.0
	S.D. 5.30	S.D. 5.77

Figure 8 illustrates graphically the relative magnitude of impedance change and eardrum displacement in Subject 3. Maximum response occurred at 105 dB SPL for both parameters of impedance change and eardrum displacement. As can be seen there seems to be a greater dynamic range for impedance than for displacement. Figures 9-12 exemplify the results obtained from this experiment. Figure 9 shows the baseline configuration for this subject. In Figure 9 no impedance changes can be seen, and no associated eardrum movement. At 10 dB higher intensity, Fig. 10 indicates a much increased impedance change but with no associated movement of the eardrum.

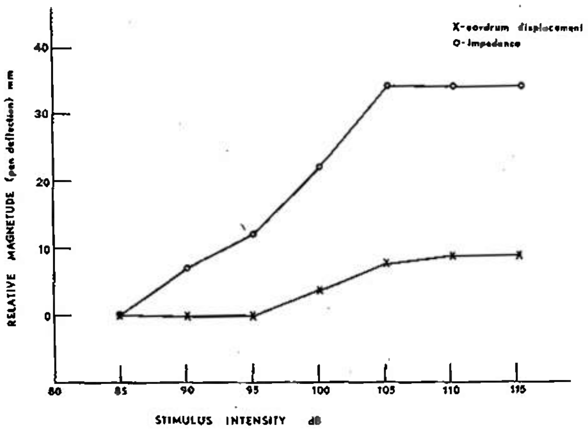

Fig. 8. The relative magnitude of eardrum displacement and impedance as a function of stimulus intensity.

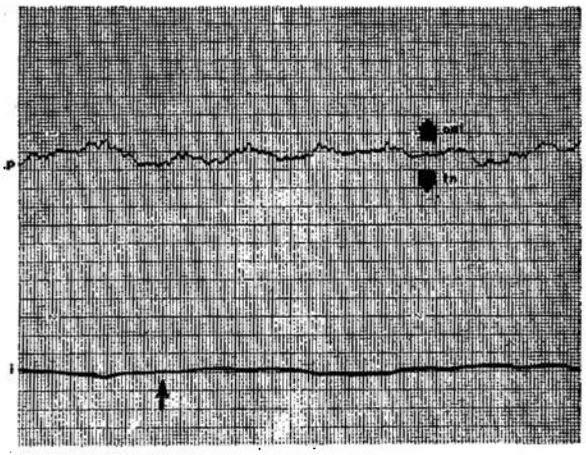

Fig. 9. Experiment I, 80 dB SPL.

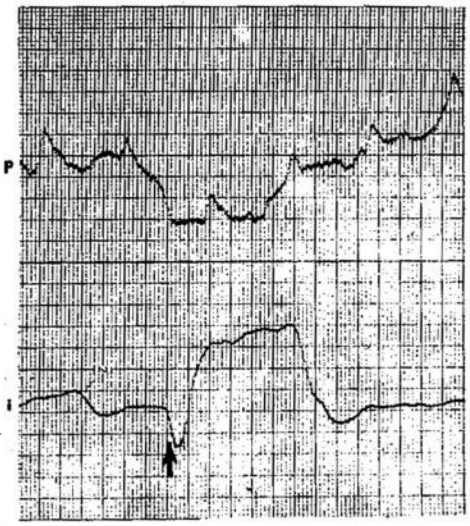

Fig. 11. Experiment I, 100 dB SPL.

Fig. 10. Experiment I, 90 dB SPL.

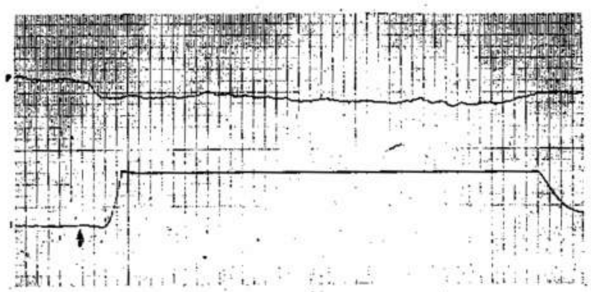

Fig. 12. Experiment I, 110 dB SPL.

With another 10 dB intensity (Fig. 11) eardrum displacement is evident with still another 10 dB intensity (Fig. 12) the very distinctive decrease in pressure (inward movement of the eardrum) follows. (In Fig. 12 the paper speed has been increased.)

If we may accept the assumption that a 5 dB above stapedius threshold the tensor is not active, it appears in these data that there is no movement of the eardrum to stapedius-only contraction. Inward movement commonly occurred, but always at higher intensities. These movements can without much question be attributed to the tensor muscle.

These results are in general concurrence with the data presented by Moller (1964)[15] and Weiss et al. (1962)[13]. These authors accepted an inward movement of the eardrum as evidence of tensor contraction. Insignificant movements or an absence of eardrum movements were found to stapedius contraction. The existance of biphasic movements as reported[14] are usually ascribed to the action of both middle ear muscles. When such biphasics are reported, quite intense acoustical stimuli are used (e.g., 127 dB used by Holst et al.).

Djupesland (1964)[25] has suggested that the tensor tympani muscle contraction may be part of the startle-related cochleopalpebral reflex. Klockhoff (1961)[20] using ears with suspected ossicular discontinuity was unable to demonstrate an acoustic reflex. However, using an air-jet to the orbit of the eye, Klockhoff demonstrated a tensor response. This response was considered to be part of the generalized startle reflex. It appears then, that an intense acoustical stimulus or other startle-inducing stimuli can produce a tensor contraction.

It is worthwhile speculating that certain biphasic responses may be entirely a result of tensor-only contraction. The resultant force vector produced by a contraction of the tensor muscle may well provide a complex motion of the eardrum which would involve a net biphasic pressure response before assuming its contracted position.

Seen in these figures, and common to all pressure measurements in the ear canal, were concommitant pulsations from the vascular system in the ear. The exact origin of these heartbeat artifacts is currently unknown. However, it has been the experience of this writer that the use of an inflatable cuff such as that included in the apparatus of Terkildsen (1957)[10] diminishes the strength of the pulses without a diminution in the sensitivity of the system to eardrum movement. This may suggest the origin to be partly in the ear canal, and the inflatable cuff may act as a cushion to these pulsations.

Experiment II: Electrocutaneous Stimulation

The response pattern, as indicated by a change in acoustical impedance, varied from subject to subject. The response was typically shorter in duration than the applied stimulus. One possible explanation would include electrode polarization. Klockhoff (1961)[20] used an electrode paste applied to the inside of the ear canal. Using this

paste, he electrically stimulated with a potential of between 2-4 volts. No electrode paste was used in this experiment and consequently a potential between 12-15 volts was needed to elicit the reflex.

Differences were noted in the maximum response amplitude before the subject reported pain. The rise-fall characteristics of the electrocutaneously elicited stapedius were typical, the onset being steeper than the cessation.

Eardrum movement responses were of three types: monophasically inward, biphasic (outward to inward), and a lack of movement. Figure 13 shows a typical monphasic-inward response from one of two subjects, responses considerably smaller than those from tensor contractions in Experiment I. Figure 14 shows a typical biphasic response from one of three subjects who exhibited this pattern. Pressure changes again are minute, of the order of a fraction of the heartbeat. On the remaining three of the eight subjects no responses could be detected with this system.

The unpredictability of eardrum movement from a stapedius contraction has been noted by Moller (1964)[15]. He states:

> *"When the stapedius muscle contracts, the eardrum movement and resultant pressure change are more difficult to predict due to the complex arrangement of the stapes and its displacement following stapedius muscle contraction. The stapedius muscle pulls the stapes in a direction which is almost perpendicular to the outward-inward movement of stapes foot plate which corresponds to a similar movement of the eardrum. Since contraction of the stapedius muscle also causes a gliding motion in the incudostapedial joint it may also produce a movement of the incus. The resultant movement of the eardrum will then depend upon the exact direction of the stapes displacement."*

The responses of the subjects shown in Figs. 15-17 indicated no movement of the eardrum to stapedius contraction.

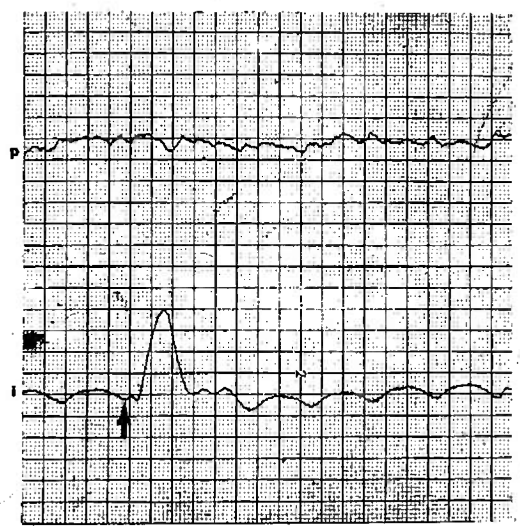

Fig. 13. Experiment II, Subject 2.

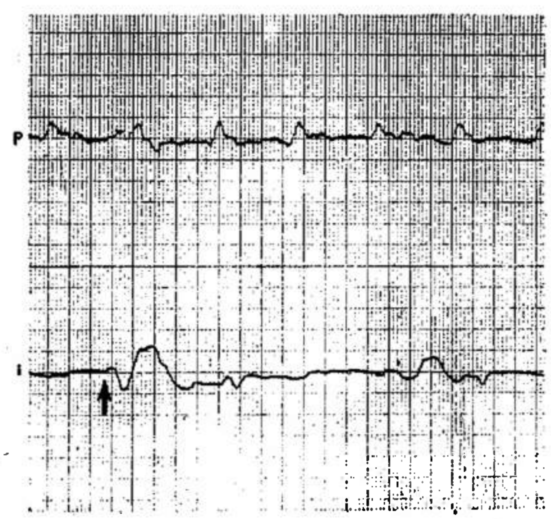

Fig. 14. Experiment II, Subject 3.

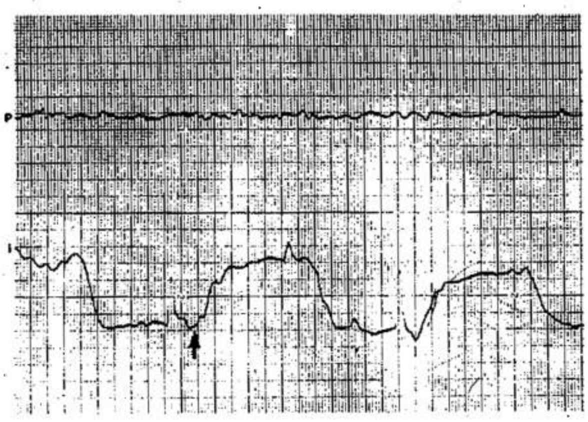

Fig. 15. Experiment II, Subject 6.

Fig. 16. Experiment II, Subject 7.

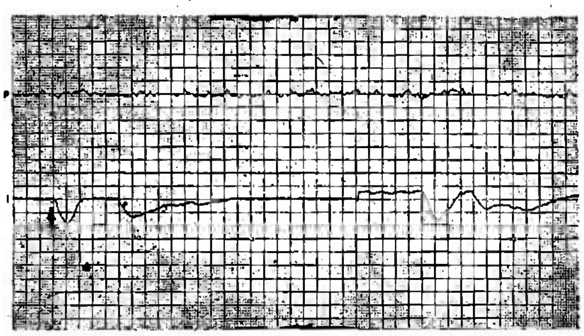

Fig. 17. Experiment II, Subject 8.

Experiment III: Normal Ears, Air-Jet to Orbit

Small biphasic eardrum movements were commonly seen in this material; they will be considered later in connection with Experiment V, the tensor-only ear.

Experiment IV: Stapedius-Only Ear Created by Cocaine Block

Three subjects submitted to anesthetization; typical set of records is summarized in Figs. 18-25. In subject 1 before anesthesia, a tensor threshold occurred at 103 dB SPL (Fig. 20) and became quite evident at higher intensities (Fig. 22). The response was distinctly indicative of inward eardrum movement and was attributed to the tensor muscle. After ten minutes of anesthetization the recordings showed a reduced response, but after 30 minutes of anesthetization (Fig. 25) note the absence of any movement of the eardrum. Eardrum movements attributable to the stapedius in this subject are not evident, though of course in all cases the impedance records show maximum stapedius contraction.

Prior to anesthetization, both biphasic movement (outward followed by the usual inward response expected from the tensor) in response to 115 dB, after complete anesthetization Figs. 26 and 27 indicate an absence of the strong inward movement attributable to tensor activity. In these subjects, however, the slight biphasic movement occurring at the beginning of the response remained, and this movement must be attributed to the stapedius muscle. Since

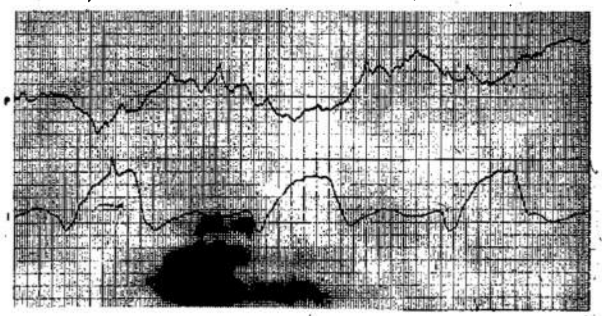

Fig. 18. Experiment IV, Subject 1, no anesthetization, 100 dB SPL.

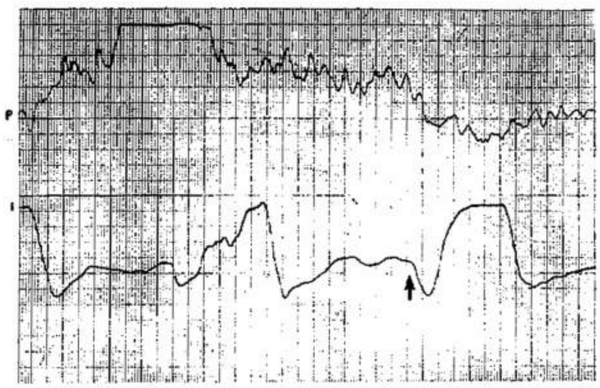

Fig. 19. Experiment IV, Subject 1, no anesthetization, 102 dB SPL.

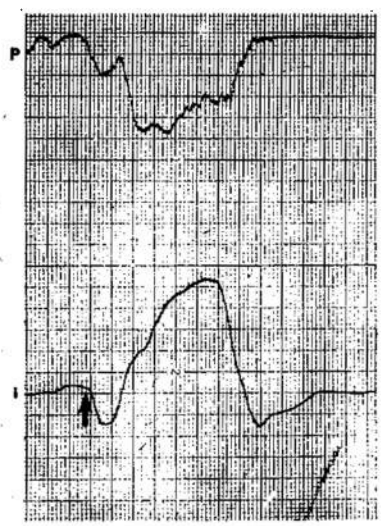

Fig. 20. Experiment IV, Subject 1, no anesthetization, 103 dB SPL.

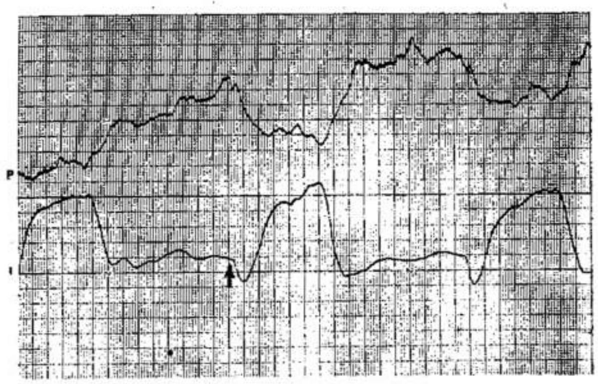

Fig. 21. Experiment IV, Subject 1, no anesthetization, 105 dB SPL.

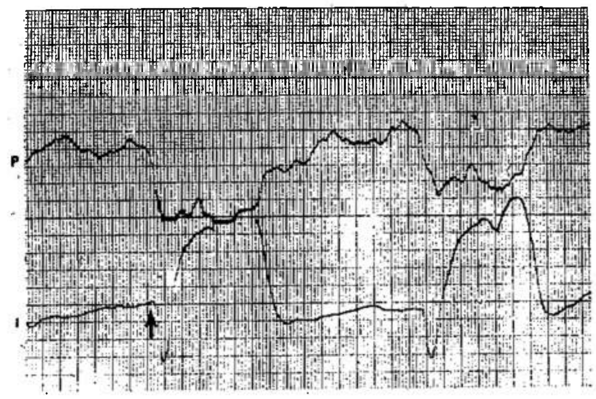

Fig. 22. Experiment IV, Subject 1, no anesthetization, 115 dB SPL.

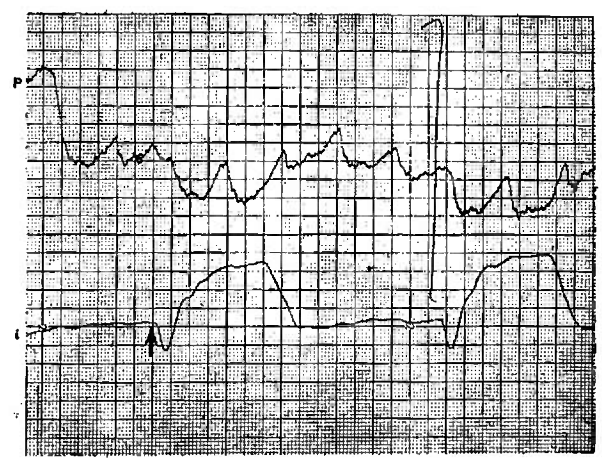

Fig. 23. Experiment IV, Subject 1, partial anesthetization, 115 dB SPL.

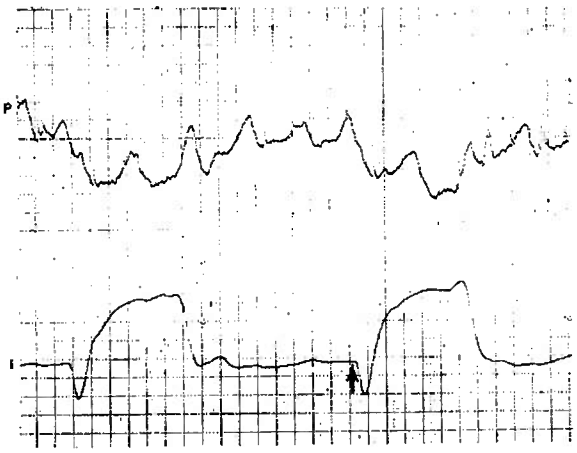

Fig. 24. Experiment IV, Subject 1, partial anesthetization, 115 dB SPL.

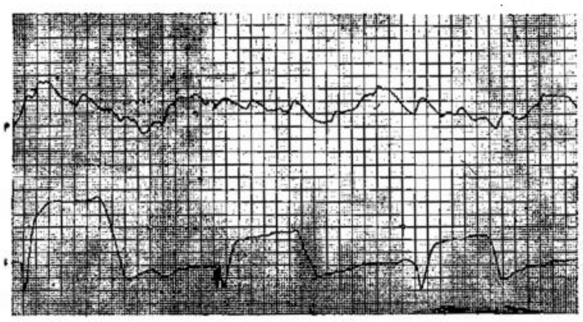

Fig. 25. Experiment IV, Subject 1, complete anesthetization 115 dB SPL.

Fig. 26. Experiment IV, Subject 2, complete anesthetization, 115 dB SPL.

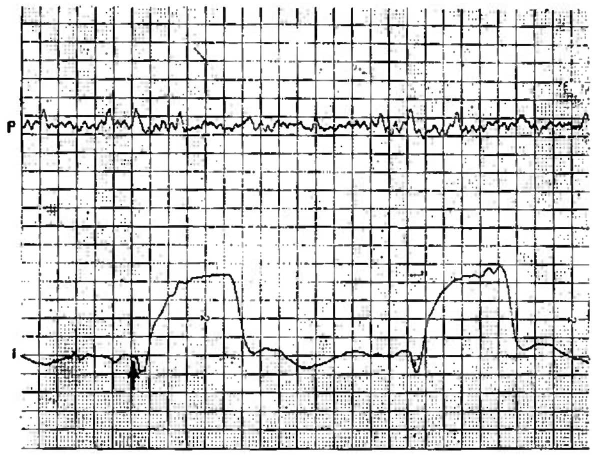

Fig. 27. Experiment IV, Subject 3, complete anesthetization, 115 dB SPL.

only 2 and 3 from this drug-blocked control experiment exhibited small biphasic movement of the eardrum to stapedius contraction, these data confirm Moller (1964)[15] that it is difficult to predict eardrum motion consequent on stapedius activity.

The lack of any outward or biphasic movement of the drum for the subjects used in Experiment I, identically stimulated, is difficult to explain. Possibly, in Experiment I the more extensive repetitions of acoustical pulses at the lower intensities (less than 110 dB) allowed adaptation of the stapedius to occur, slight but sufficient to eliminate its ability to cause eardrum displacement. Detailed adaptation studies of the stapedius muscle are currently needed.

Experiment V: Tensor-Only Ear (Stapedectomy)

Any movements which occurred in the previous experiments attributable to

the stapedius muscle would necessarily be absent in this group.

Audiograms are shown in Figs. 28 and 29. In Subject 1, both ears lacked a stapedius muscle. Subject 2 lacked a stapedius muscle in the left ear. The loss in the right ear was also due to otosclerosis and another operation was anticipated by this subject.

Only Subject 1 indicated eardrum movement to an auditory stimulus (1 kHz, 115 dB SPL). Fig. 30 indicates a very slight change in the acoustical impedance, but a substantial monophasic inward movement of the eardrum is quite evident. It was only at this high acoustic intensity that a response was obtained, and only in the left ear. No electrocutaneous stapedius response was obtained with either subject; considerable validity must therefore be assigned to the strong electrocutaneously-elicited stapedius response in the normal-hearing subjects of Experiment II.

An air-jet to the orbit of the eye was applied to each stapedectomized subject. The results were very dramatic as compared to the results with this stimulus of the normal-hearing subjects of Experiment III. Very strong entirely inward movements of the eardrum are seen in Figs. 31, 32. Oftentimes this occurred with a concurrent impedance change (see Fig. 33 for a detailed example). This latter may be monophasic (Fig. 34) or biphasic (Fig. 27).

We here compare these rather extreme monophasic eardrum movements

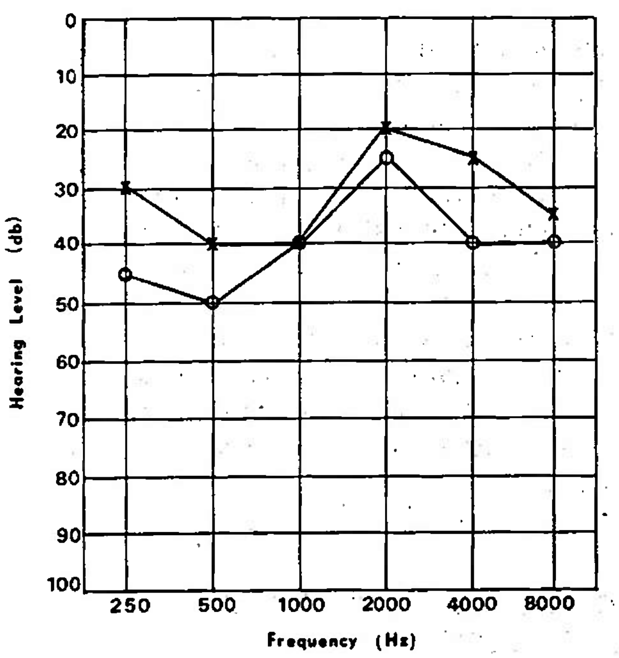

Fig. 28. Experiment V, Subject 1 audiogram.

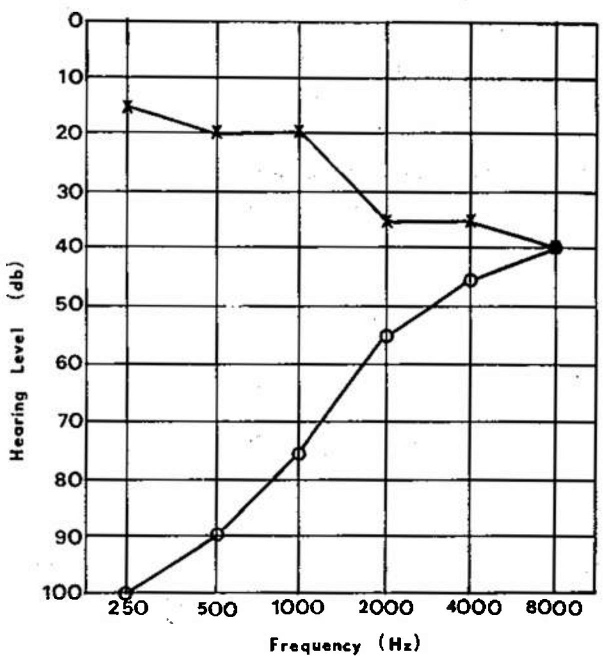

Fig. 29. Experiment V, Subject 2, audiogram.

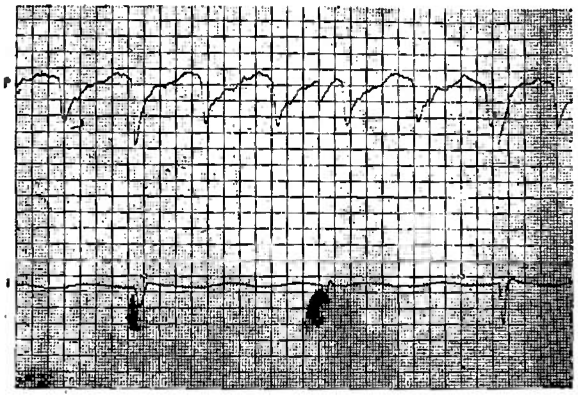

Fig. 30. Experiment V, Subject 1, acoustical stimulation, 115 dB SPL.

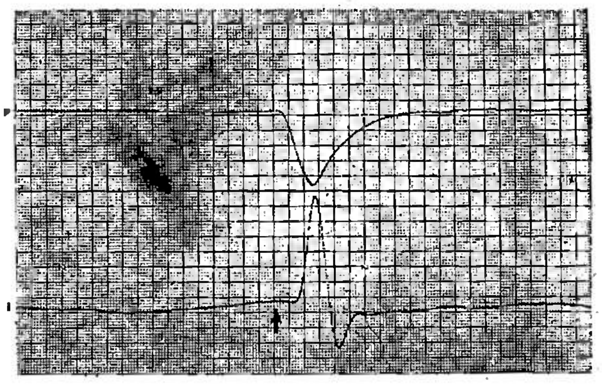

Fig. 33. Experiment V, Subject 2, air-jet stimulation.

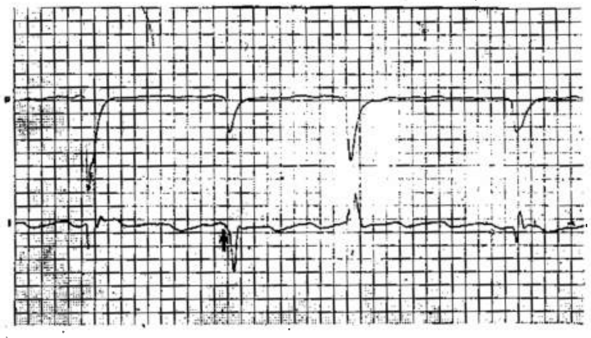

Fig. 31. Experiment V, Subject 2, air-jet stimulation.

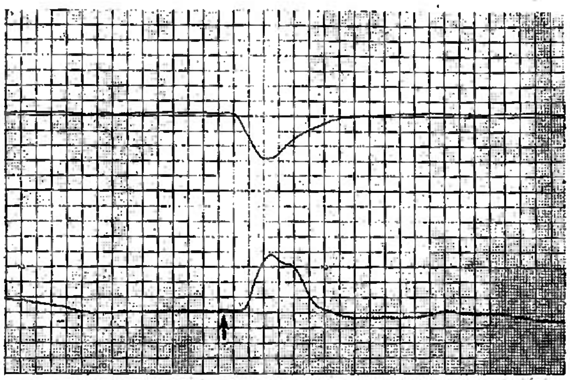

Fig. 34. Experiment V, Subject 2, air-jet stimulation.

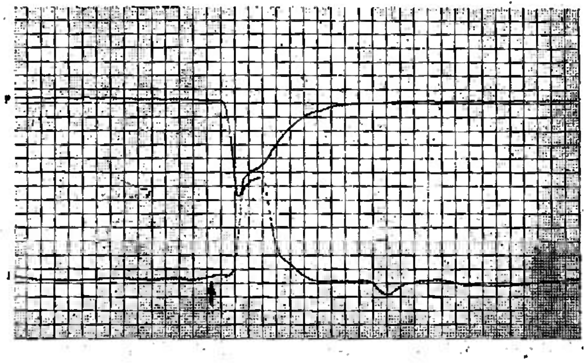

Fig. 32. Experiment V, Subject 2, air-jet stimulation.

to orbital stimulation of these tensor-only ears with the small biphasic eardrum movements to air-jets in the normal material of Exper. III (see Figs. 35, 36). It seems that a lack of the stapedius muscle allows accentuation of the tensor's effect on the eardrum. The suggestion here appears plausible that the stapedius with its associated tonus may in some way modulate tensor contraction.

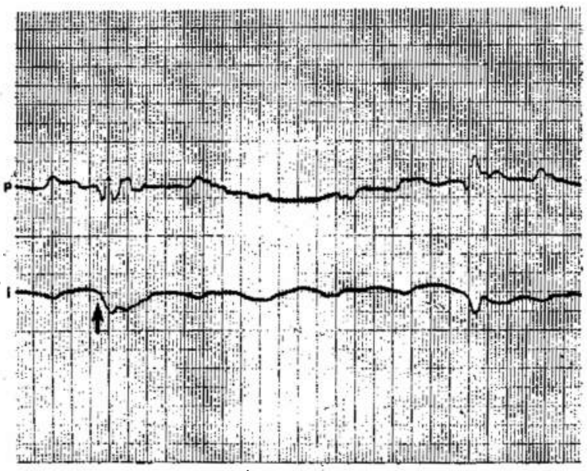

Fig. 35. Normal ear, air-jet stimulation.

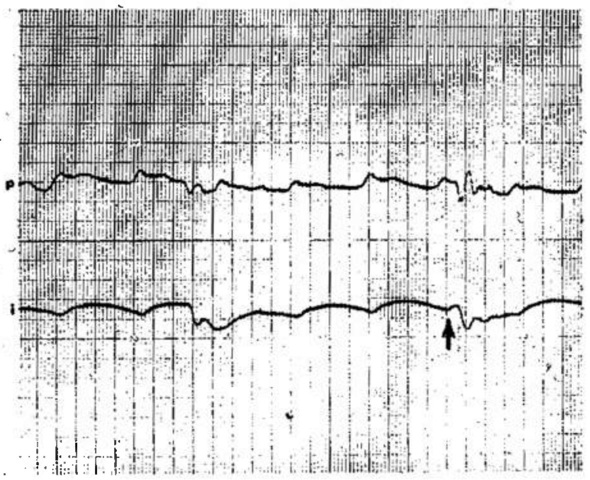

Fig. 36. Normal ear, air-jet stimulation.

Model to Estimate Eardrum Displacement

In this section, a model of the ear canal, including the eardrum, was formulated in order to determine the actual displacement of the eardrum. Fig. 37a describes the closed cavity formed by the eardrum, ear canal, and the sealed apparatus including the manometer membrane. The total volume of this closed cavity is represented by V.

To determine the displacement of the tympanic membrane the enclosed volume V is expressed in terms of pressure, P, by the equation of state[26]:

$$PV = RT \tag{1}$$

where P is the pressure enclosed in V. R is the universal gas constant, and T is the temperature.

Differentiating (1) gives:

$$PdV + VdP = RdT \tag{2}$$

Assuming the temperature to remain constant when the pressure, P, in volume V, changes, equation (2) can be rewritten as,

$$PdV = -VdP \tag{3}$$

Since $|P_1 - P_2| = |P_2 - P_1|$, (3) can be written as,

$$PdV = VdP \tag{4}$$

Substituting delta (Δ) notation for the derivatives, equation (4) becomes:

$$P\Delta V = V\Delta P \text{ or } \Delta V = \frac{V\Delta P}{P} \tag{5}$$

Thus, equation (5) expresses the volume displacement, ΔV in terms of P, at-

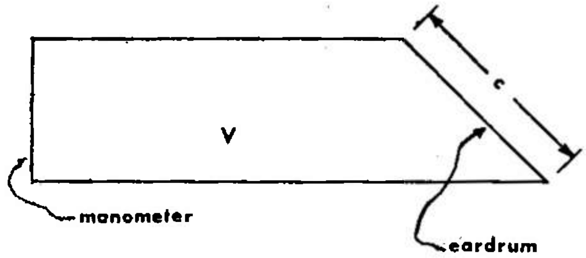

Fig. 37a. Closed cavity formed by the eardrum, ear canal, and sealed apparatus.

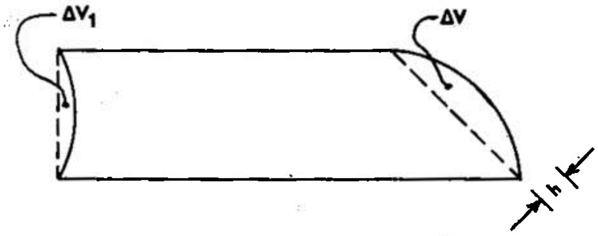

Fig. 37b. Assumed model of the motion of the eardrum and manometer membrane.

mospheric pressure; ΔP the change in pressure in the closed cavity and V, the total volume of the cavity.

Fig. 37b illustrates the motions assumed for the manometer membrane and the eardrum. The eardrum was assumed to move and displace as a segment of the volume of a sphere. The volume it would displace is ΔV. Knowing C, the length across the eardrum (average of the minor and major axis), h, the actual displacement can be found from the mensuration formula of the volume of the segment of a sphere[27]. Equation (6) describes this formula:

$$\Delta V = h^2 \pi \left(\frac{C^2 + 4h^2}{8h} - \frac{h}{3} \right) \quad (6)$$

A correction to ΔV must also be made. The volume displacement, ΔV_1 caused by the consequent movement of the manometer membrane is given from the specifications supplied by the transducer manufacturer. The corrected volume displacement can now be expressed as:

$$\Delta V = \Delta V - \Delta V_1 \quad (7)$$

The empirical values needed to obtain ΔV are P, ΔP, and V. P was obtained from a mercury barometer and converted to mm of H_2O. The average ΔP attributed to the tensor muscle was found to be .02 mm of H_2O. The average V was empirically determined by dispensing alcohol from a syringe through the entire closed system. V was measured to 14 cc or 14,000 cu mm.

Substituting these values into equation (5) and (7) gives:

$$\Delta V = .01377$$

The value of C, the distance across the eardrum, was taken to be 9 mm (Wever and Lawrence, 1954).[1] The eardrum was assumed to be circular.

SUMMARY AND CONCLUSIONS

The major purpose of this study was to investigate eardrum movement during stapedius muscle contraction. A direct measure of air pressure changes in the external auditory canal provided information concerning eardrum displacements. Simultaneously, on the

same ear an electroacoustic impedance bridge recorded the characteristic impedance of the eardrum.

Control procedures were established in order to elicit only a stapedius contraction, and to test the validity of associated displacements of the eardrum. To elicit only the stapedius muscle the following procedures were employed:

(1) Acoustic stimuli consisting of a 1-kHz pure tone were presented, and increased from 70 dB to 115 dB SPL with increments of five dB.

(2) An electrocutaneous stapedius reflex was elicited using a surface electrode in the external ear canal.

(3) Blocking of the tensor muscle was accomplished through anesthetization of the sphenopalatine ganglion.

As a means of testing the validity of movements associated with the stapedius muscle and to examine the responses of the tensor-only ear the following procedures were employed:

(1) Patients who had recently undergone stapedectomy were subjected to stimuli that were identical to those presented acoustically, electrocutaneously, and tactually from a normal ear.

Extensive data were collected from 22 subjects. The following observations and conclusions were made:

(1) Using as stimulus a 1-kHz tone, eardrum displacement occurred only when intensity reached about 100.0 dB SPL, although simultaneous impedance measurements always indicated a strongly active stapedius reflex in all eight subjects at much lower intensities. Movement of the eardrum was found in seven of these eight, always monophasically inward; this movement was unequivically attributed to the tensor muscle. In one of the eight, no movements were observed.

(2) Electrocutaneous stimulation was successfully employed in eight subjects to elicit stapedius-only contraction. Small monophasic inward movements of the eardrum were observed in two subjects, and small biphasic movements (outward to inward) of the eardrum were observed in three; all of these movements were attributed to the stapedius muscle. No movement was found in the remaining three subjects.

(3) Anesthetization of the sphenopalatine ganglion was used successfully in three subjects to block tensor contractions. In response to sound, small biphasic (outward to inward) movements attributed to the stapedius muscle, were observed in two subjects. In one subject there were no movements.

(4) Three ears from two subjects that lacked a stapedius muscle exhibited no eardrum movement of any kind to acoustic stimulation at up to 115 dB SPL, or to homolateral electrocutaneous stimulation. These same subjects, however, when stimulated with an air current to the orbit of the eye exhibited a stronger than normal inward-only displacement of the eardrum; this response contrasts to the weak biphasic response often seen in a separate group of three normal subjects given air-jet orbital stimulation. From the literature, one would assume that these three normal-

hearing subjects were exhibiting tensor-only response. In this material, also, simultaneous impedance recording indicated only very slight impedance increase which can only be attributed to eardrum displacement, consequent upon tensor-only contraction. This displacement, predominantly inward, is modified by tonicity or contraction of the stapedius muscle if the latter is in fact present.

(5) An approximate measurement of the actual displacement of the eardrum was calculated from a modeled eardrum and canal. This displacement amounted to 4.1 x 10^{-4} mm.

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13. ABSTRACT

Simultaneous monitoring in human subjects on the same ear of eardrum displacement by tympanomanometry, and acoustical impedance by the Madsen bridge, provided information concerning contraction of the stapedius muscle and its effect on eardrum displacement. Extensive control procedures were employed to elicit only the stapedius: lower-intensity auditory stimulation, electrocutaneous stimulation of the homolateral external ear canal, and anesthetization of nerves leading to the tensor tympani. 1. Extremely small biphasic and monophasic eardrum movements were seen in the stapedius-only ear to auditory and electrocutaneous stimulation; the form of the response was much less predictable to auditory stimulation. 2. At higher sound intensities relatively large inward and biphasic movements of the eardrum occurred in the normal ear, unquestionably due to contraction of the tensor tympani. These results were further validated in a group of stapedectomized ears, without the stapedius but with normal tensor tympani. 3. Biphasic responses did not occur in the tensor tympani-only ear only monophasic inward responses. 4. Upon air-jet stimulation to the orbit of the eye, these subjects had an accentuated tensor response in that large inward movements of the eardrum occurred as compared to those in normal ears, suggesting that there is an alteration of the tensor response by the presence of the stapedius muscle. Estimates of the actual eardrum displacements were calculated based on a model of the external ear canal and eardrum.

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